FALSE DIVERTICULA OF THE VERMIFORM APPENDIX.

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DIVERTICULA of the intestinal tract are classed as true or false, this classification being based on the histological structure of the wall of the diverticulum. If the wall is made up of all the normal coats of the intestine the term true diverticulum is used, as in the case of Meckel's diverticulum. If the muscular coat is lacking wholly or in part, it is called a false diverticulum. The true diverticula are practically always congenital, whereas the false ones are usually acquired. These facts all hold good for the appendix, in common with the rest of the intestinal tract, although there is no record in literature of an undisputed case of a true diverticulum of the vermiform appendix.

Considering the serious clinical import of these pathological structures, it is rather anomalous that they have received such scanty attention in our voluminous literature on diseases of the appendix. In American publications no reference to them has ever been made.[1] Kelly makes no mention of them in his encyclopedic work on the appendix; in the foreign journals only two articles have been written which deal exclusively with diverticula of the appendix, and only six or eight, all told, have been written, which treat of the subject, in connection with intestinal diverticula in general.

Practically all surgeons are agreed on the impossibility of establishing a correct anatomical diagnosis in the presence of appendicular disease. If it can be shown that serious involvement of this organ predisposes to diverticula formation, and that these pathological pouches are a menace to life, then another advance has been made in our attempts to link the pathological with the clinical aspect of appendicitis.

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[1] This statement will have to be modified, for since this paper was finished I found a reference (Cent. f. Chir. No. 7, 1906) to an article by Corning in the *Albany Medical Annals* for December, 1905, entitled Retention Cyst and Diverticulum of the Vermiform Appendix.

Through the kindness of Dr. Harvey Mudd I obtained an appendix which showed one large fully-formed diverticulum, and a small diverticulum in process of formation. The organ (Fig. 1) was removed by Dr. Mudd during the course of an abdominal hysterectomy for uterine fibroids. The patient gave no definite history pointing to an acute attack of appendicitis, having suffered only from pelvic pain and the other classical symptoms of fibroids. The appendix, which measured 7 cm. in length, was covered by a glistening non-congested serosa; 1 cm. from the distal end was a diverticulum measuring 8 mm. across the base by 4 mm. in height (Fig. 1 d); 2½ cm. from the end was a small nodule (Fig. 1 d2)) which presented all the macroscopical appearances of the fibrous nodules so often encountered on the peritoneum, but which on microscopical examination proved to be the protrusion of the submucosa through a hiatus in the musculature. Both of these diverticula were situated midway between the mesenteriolum and the free convex border of the appendix. The lumen of the appendix was injected with 80-per cent. alcohol, the proximal end being tied off while the organ was distended, in order to preserve the contour of the diverticulum during the process of fixation. Serial sections were then made through the nodule d2 and the diverticulum d. Each series embraced healthy tissue above and below these structures.

Fig. 2 shows a cross section through the large, fully-developed diverticulum represented as in Fig. 1. The muscular coat is broken in two places,—at b, where a normal break always occurs to admit the mesenteric blood-vessels, and at x-x, where the mucosa and submucosa protrude to form the diverticulum. The break in the muscularis caused by the entrance of the mesenteric blood-vessels is filled in by the vessels themselves, and by perivascular connective tissue. Yet, despite this fact, the broken continuity creates a locus minoris resistentiæ at this point, which, as a result, constitutes a site of predilection for diverticulum formation. In the present case, however, the diverticulum did not form at this point of natural weakness, but at a spot almost directly opposite to the mesenteric attachment. The ends of the muscular coat thin down and terminate abruptly at x-x, as if they had been trimmed with a knife. Sections taken at a different level, however, show a somewhat different picture, in so far as the two ends of the muscle-crescent do not terminate abruptly, but are prolonged as thin fibres, into the wall of the diverticulum, along a part of its circumference. The muscular coat in Fig. 3, which represents a section through d2 Fig. 1, shows a loss of substance similar to that seen in Fig. 2. Aside from this loss of substance, there is no other pathological change in the muscle; no fragmentation, fatty infiltration or degeneration, and no nuclear changes. The structure of the submucosa varies in no way from the normal structure of this coat, except that it protrudes between the ends of the muscle-crescent to form part of the wall of the diverticulum in Fig. 2, and to form the subperitoneal nodule in Fig. 3. The mucous membrane in both sections is also normal as regards the distribution of the lymphoid tissue, the size, shape and number of crypts, and the character of the epithelium lining these crypts. The lumen of the appendix (l, Figs. 2 and 3) is not situated centrally as it should be, but has been forced into an excentric position by the protrusion of the coats. This excentricity of the lumen is a characteristic result of diverticulum formation. Fig. 4 is a schematic representation of the development of this displacement of the lumen. The uppermost drawing of Fig. 4 shows a section taken just above the small nodule d2 (Fig. 1). The lumen is centrally situated. As the sections proceed downward toward the tip of the appendix, they pass through the nodule, showing both the bulging of submucosa through the muscularis, and the displacement of the lumen. This excentricity is, of course, due only to the thinning out of the appendicular wall in one place and the thickening of it in another.

The wall of the diverticulum itself is made up throughout two-thirds of its extent by mucosa, submucosa and serosa (Fig. 2). There is an entire absence of muscularis, except where the horns of the muscle-crescent embrace the proximal part of the diverticulum. The submucosa does not differ in appearance from normal submucosa; but the mucous membrane lining the diverticulum varies markedly from normal appendicular mucous membrane, in that the lymphoid tissue is more sparsely distributed, the crypts less numerous, and more irregular in shape, and the epithelium lining them of low cuboidal instead of high cylindrical shape. Similar changes in the mucosa lining the diverticulum have been described by V. Brunn[2] and by Lejars and Ménétrier,[4] authors who refer these changes to the existence of a past inflammation, which, in their opinion, accounts for the formation of most diverticula of the appendix. There is an absence of muscularis mucosæ both in the wall of the appendix and in the wall of the diverticulum. Obendorfer[11] states that absence of the muscularis mucosæ points to the fact that the appendix was the site of a chronic inflammation.

The Site of Diverticula of the Appendix.—By far the largest proportion of the fifteen or twenty diverticula of the appendix recorded in literature, occurred at the mesenteric attachment of the organ. This fact is readily understood, after a glance at Figs. 2, 3, and 4, which show how the continuity of the muscularis is interrupted, in order to afford entrance to the nutrient blood-vessels. As already stated, this break in muscular continuity causes a locus minoris resistentiæ, and allows the submucosa and mucosa to be herniated through the weak spot, to form a diverticulum lying between the leaves of the mesentery. The muscular coat is preëminently the strong coat of the appendix. Oschner[1] has stated that no other tube in the body possesses so thick a muscularis, in proportion to its size, as does the appendix. The submucosa is strong by reason of its passive opposition to the intra-appendicular pressure, but the muscularis, in addition to the passive opposition afforded by its thickness, exerts active opposition through its contractile power. Naturally, therefore, that spot where the muscularis is lacking, will be a weak spot, and one through which a hernial protrusion is most likely to occur.

But the mesenteric border is not the only site at which the muscularis may be lacking. Defects may occur in any part of the muscularis, and as a result, diverticula are reported by von Brunn,[2] Edel[3] and Lejars and Ménétrier[4] as occurring in the neighborhood of or opposite to the mesenteric border of the appendix. The appendix dealt with in this paper showed an intact mesenteric border, with hernial protrusions near the opposite side. As regards situation, therefore, diverticula occur most frequently at the mesenteric border, but they may occur also at any point on the circumference of the appendix.

The Etiology of Diverticula of the Appendix.—Two factors are essential for the development of these diverticula, a weak spot in the wall of the appendix, and pressure within the appendicular lumen sufficient to cause the mucosa to be forced outward through the weakened wall. Both of these factors are present in every normal appendix; for there is always a positive intra-appendicular pressure greater than the negative intra-abdominal pressure: and the muscularis of the appendix is always broken by the entrance into and passage through it of the nutrient vessels, coming from the mesenteriolum. The fact that the muscularis is weakened by the blood-vessels coursing through it has been demonstrated experimentally by several authorities. Heschl[5] removed pieces of intestine from the body, and distended them with water. He found that after the intestine was distended, small points of bulging could be seen along the mesenteric border. These artificially-created diverticula disappeared as the water was let out. Hansemann[6] confirmed Heschl's observations, and Grassberger[7] independently reached the same conclusions.

It is only natural to suppose, then, that since the mesenteric border of the appendix is naturally a locus minoris resistentiæ, and that since there is always a positive pressure present in the appendix, we should expect diverticula to occur most frequently along the line of mesenteric attachment. This supposition is confirmed by the list of cases already recorded in literature. Yet there is an important group of cases in which the diverticula do not form along the mesenteric attachment, but along the convex border of the appendix. Here, as in the former cases, the two factors of intra-appendicular pressure, plus a weak spot in the appendix wall, are present; but the weak spot in the wall is an acquired one, the result of past inflammation. The patient may have been the subject of a very acute appendicitis which resulted in perforation, or he may have suffered from a less severe attack, without perforation. In either case, however, the appendix wall was destroyed at one point (by a process of necrosis in the one case, or by a dense round-cell infiltration in the other). The mucosa of the appendix, in common with the mucosa of all other organs, possesses the power of regeneration. The submucosa and the endothelial cells of the serosa also manifest this same property. The muscularis, however, being one of the most highly-evolved tissues of the body, cannot regenerate. The result of all this is, that after a severe acute attack of appendicitis the involved portion of the wall of the appendix shows a regenerated mucosa and submucosa, and a muscularis in which the parenchyma has been replaced by connective tissue. While this connective tissue is young it is yielding and plastic, and being thus weaker than the normal muscularis, it gives way before the intra-appendicular pressure behind it and thus permits a diverticulum to form.

Although the two factors—a weak wall and pressure from within—are constantly present, we see nevertheless that there is the third important factor, inflammation, to take into account when considering the etiology of diverticulum formation. A previous inflammation of the appendix not only furnishes a locus minoris resistentiæ, but determines where the diverticulum will form. If the convex border of the appendix was the site chiefly involved, a diverticulum may form here; if the mesenteric border was chiefly involved, a diverticulum may form at this site, which, though naturally a weak spot, was nevertheless strong enough to resist diverticulum formation until compromised by the acute inflammatory attack.

THE CLINICAL SIGNIFICANCE OF DIVERTICULA OF THE APPENDIX.

Diverticula of the appendix are of the most serious clinical import; and it is only due to the fact that they have up to now been regarded as pathological curiosities, that their true clinical significance has been overlooked. Undoubtedly the most dreaded outcome of appendicitis is perforation, with consequent purulent peritonitis. The diverticulum furnishes the most favorable set of conditions for perforation. A thin-walled sac with no muscularis in its make up, opening into the appendicular lumen, through a mouth that is always small, and therefore easily closed off by swelling of the mucosa or by a plug of feces, must be a highly dangerous intra-abdominal content. Of course, after an appendix has perforated it is rather fruitless to attempt to show whether the perforation occurred at the site of a former diverticulum or not. Von Brunn[9] and Mertens[10] both assert the belief that cases of perforation under their observation were referable to diverticulum formation. Even granting that a diverticulum does not perforate, it is still a menace, for Helmberger and Martina[8] have shown that it is chiefly the muscular coat of the intestine which opposes the migration of bacteria from within; and in diverticula, the muscular coat is absent.

There is no small amount of significance in the clinical fact that in the large majority of foudroyant cases of appendicitis there is a previous history of repeated acute and subacute attacks, terminating finally in a particularly stormy attack with peritonitis. In our own case, there was no definite history of appendicitis; yet it is very probable that an attack of pain, in this particular individual, would have been referred to the presence of the large fibroid uterus. The increment in severity which marks the successive attacks of appendicitis may be fairly assumed to depend upon the damaged condition of the appendix. Since it has already been shown how this damage may result in diverticulum formation, the clinical

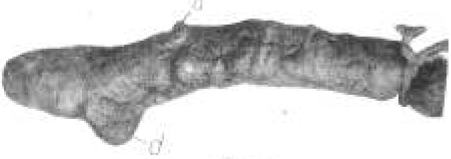

Fig. 1

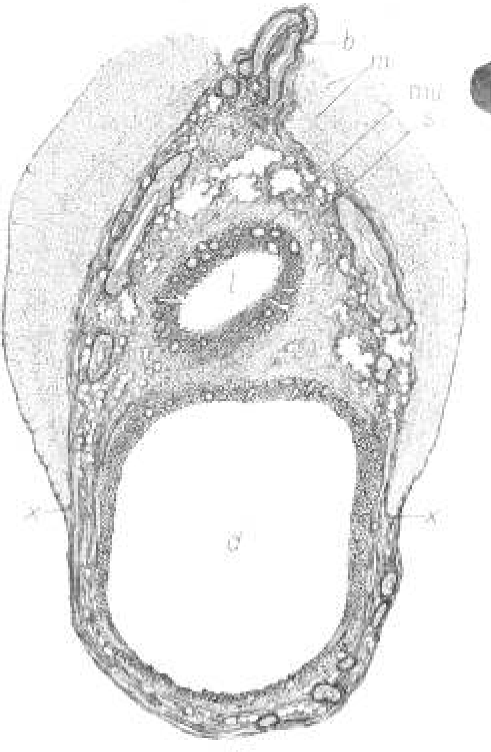

Fig. 2

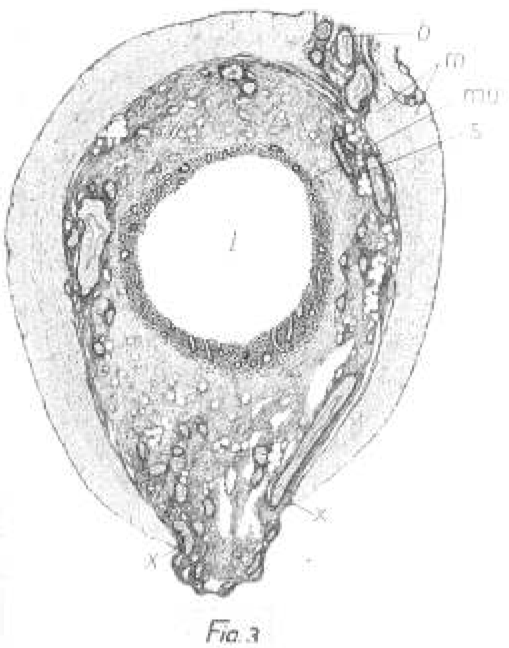

Fig. 3

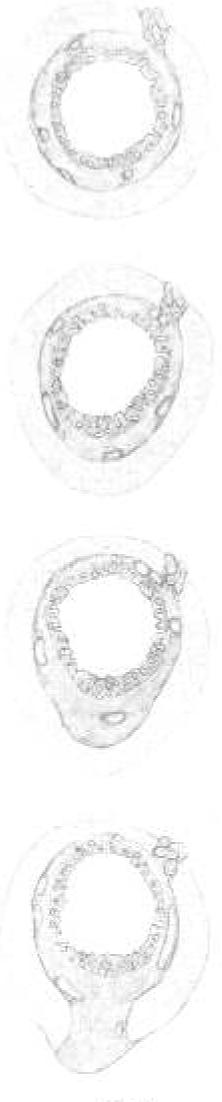

Fig. 4

significance of these structures becomes apparent without further comment.

It must be borne in mind, of course, that a somewhat differing significance attaches itself to diverticula according to whether they have formed idiopathically, through the weak cleft caused by the vessels entering the muscularis, or whether they have resulted from past acute inflammation. Concerning diverticula which protrude between the leaves of the mesentery, we can only say that they are a constant menace, but that their presence is unsuspected until, owing to the thinness of their wall, infection travels through them or perforation occurs. The diverticula that result from inflammation, however, are of more significance to us, in that a knowledge of the possibility of their existence enables us to set a very definite indication for operation in those cases of appendicitis after an acute attack. At all events they teach us that there are excellent pathological reasons for anticipating serious trouble from the appendix which has once been markedly compromised by inflammatory disease.

EXPLANATION OF PLATE.

Figure 1.—Photograph of appendix enlarged one-third. d, fully formed diverticulum; d^2, small nodule formed by protrusion of submucosa.

Figure 2.—Section through d (Fig. 1). b, blood-vessels from mesenteriolum; m, muscularis; mu, mucosa; s, submucosa; x-x interruption in continuity of muscularis; l, lumen of appendix; d, lumen of diverticulum.

Figure 3.—Same as Fig. 2.

Figure 4.—Gradual displacement of lumen of the appendix to an excentric position as a result of protrusion of the submucosa.

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